low-potency steroids had no effect. Our patient was treated with a modern glucocorticoid which has an improved risk–benefit ratio. The antipruritic and anti-inflammatory properties of the steroid were increased by applying it in combination with a wet-wrap technique, which has already been shown to be extremely helpful in cases of acute exacerbations of atopic eczema in combination with (3) or even without topical steroids (8).

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Autoinvolutive Photoexacerbated Tinea Corporis Mimicking a Subacute Cutaneous Lupus Erythematosus

Sir,

Occasionally, tinea corporis may resemble different conditions, including lupus erythematosus (LE), psoriasis, pityriasis rosea, nummular eczema or annular secondary syphilis. Its misdiagnosis constitutes a problem of considerable practical importance. We describe here a patient who presented with a widespread cutaneous eruption, which reappeared at regular intervals after sun exposure. The morphology and distribution of the lesions mimicked a subacute cutaneous lupus erythematosus (SCLE).

CASE REPORT

A 69-year-old white man, first seen in September 1999, presented with a 3-month history of an asymptomatic extensive eruption constituted by well-defined annular erythematous patches and plaques which covered, in a symmetrical pattern, his whole back and the dorsal aspects of his arms (Fig. 1). Further physical examination, including the feet and nails, was unremarkable. No systemic signs or symptoms were present. The personal history was not relevant except for an autoimmune hypothyroidism. His history dated back to June 1996, when the patient noticed a flare of lesions similar to those described above. The patient related that the lesions had appeared in summer after sun exposure, and had completely faded in the autumn without treatment. New outbreaks with the same course had reappeared during the next three summers. A clinical diagnosis of SCLE was made.

The following measurements were negative or within normal limits: blood cell count, biochemical and urine analysis, serum complement, immunoglobulins, peripheral lymphoid subpopulations, antibodies against DNA, SS-A/Ro, SS-B/La, Sm, and RNP and human immunodeficiency virus (HIV) serology. The ANA titre was 1:20. A skin biopsy showed hyperkeratosis with parakeratosis, a discrete vacular degeneration of the basal layer, and numerous hyphae in the stratum corneum and hair follicles. Direct immunofluorescence was negative. KOH examination of a skin scraping taken from the back revealed abundant branching hyphae. Fungal culture grew Trichophyton rubrum. There was no known underlying disease or immunosuppression that could favour a dermatophyte infection. In the 2–4 weeks after his first visit, the lesions improved greatly and spontaneously. A very slight erythema and scaling remained in some areas, while there was apparently complete healing in other areas. There were also scarce follicular pustules. The patient was treated for 2 months with oral terbinafine and topical tioconazole, during which time the lesions were cured. A control biopsy showed no relevant findings. The skin has remained normal for one year, including last summer.

DISCUSSION

Sometimes, dermatophyte infections show an atypical pattern that simulates different skin diseases. The misdiagnosis of tinea mimicking LE has been reported in the literature. The great majority of cases are tinea faciale, with only some exceptions...
involving the trunk (1–3). Most of the cases are discoid LE-and systemic LE-like eruptions. The dermatophytes responsible for such infections are varied and include \textit{T. rubrum}, \textit{T. mentagrophytes}, \textit{T. tonsurans}, \textit{T. verrucosum} and \textit{M. canis}. Some cases coexist with true LE (1, 2). Previous treatment with topical corticosteroids might justify some patients being erroneously considered as having LE. In our case, the clinical morphology and the photoinduction of the lesions led us to consider a diagnosis of SCLE. The onset of a dermatophyte infection after a blistering sunburn, the photoexacerbation of the eruption and the presence of lesions mainly confined to sun-exposed areas are events that have been rarely reported in the literature (2–4). The influence of a suitable environment (warmth, humidity) on host skin during the summer should be considered. Widespread tinea corporis has been described in immunocompromised patients. However, in our patient there was no known immunosuppression that could favour the spreading of the lesions.

We should emphasize the apparent spontaneous resolution of the lesions and their reappearance at regular intervals in the same anatomical sites. The existence of breakdowns in the local immune response, which reflects the relatively immuno-deficiency of some areas (5), has been postulated as an explanation for this rarely described phenomenon. However, experimental studies have shown that recently affected sites are harder to infect (6). Another more convincing explanation for this peculiar course of the disease focuses on the hair follicles. These could act as a reservoir for the fungus and thus lead to a recurrence of the disease. The presence of hyphae in the follicles of our patient, and the observation of scattered follicular pustules in his back after the spontaneous apparent disappearance of the eruption, support the role of hair follicles as a reservoir for the infection.

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